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Pathways to Schizophrenic Psychosis: A LISREL-Tested Model of the Unfolding of the Schizophrenic Prodrome

▼
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In this article a literature-based model (the Schizotypic Syndrome Questionnaire [SSQ] model) is presented that gives a description of the temporal unfolding of the schizophrenic prodrome. As a guiding principle for the selection of the symptoms in the model, the hypothesis was held that the main prodromal features determine each other in terms of cause and effect. Furthermore, the developmental pathways between the symptoms were not allowed to be in conflict with the usual observation that negative symptoms precede psychotic-like ones nor—at least in broad outline—with J.P. Docherty, D.P. van Kammen, S.G. Siris, and S.R. Marder's (1978) description of the various onset stages in the development of a schizophrenic psychosis. For the definitive version of the SSQ model, 12 symptoms were selected (e.g., affective flattening, suspicion, and delusional thinking). After specifying the paths to be estimated, the model was examined in two randomly drawn samples from a total community-based sample of 771 normal subjects and in the total sample itself, in each case resulting in adequate fit values. Moreover, all postulated pathways were found to be significantly different from zero. The use of a normal sample was based on the continuum hypothesis. Given the present-day discussions concerning the tenability of the schizophrenia concept, the model's implications with respect to that issue are particularly emphasized. Furthermore, the concept of the schizophrenia prodrome itself is critically discussed. © 2005 Wiley Periodicals, Inc. *J Clin Psychol* 61: 909–938, 2005.

Keywords: schizophrenia; schizophrenia prodrome; Schizotypic Syndrome Questionnaire; LISREL-tested model; onset stages; negative symptoms; positive symptoms; psychotic-like symptoms; decompensation; schizotypy

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Introduction

In medicine, the term *prodrome*, derived from the Greek *προδρομος* (meaning *fore-runner*), denotes a constellation of symptoms that is present prior to the emergence of the characteristic symptoms of a disease. Applied to schizophrenia, the initial prodrome has been defined as a period of prepsychotic disturbance ranging from the onset of the first noticeable features to the onset of frank psychotic phenomena, particularly delusions and hallucinations (e.g., Loebel et al., 1992; Yung & McGorry, 1996a). The dating of the prodromal and the psychotic onset may be assessed by the patient himself or by a significant other, although these judgments do not necessarily lead to the establishment of the same time points or interval. Moreover, the estimation of the beginning of the prodrome is especially difficult, due to the insidious onset of the early emerging symptoms. In contrast, the establishment of the end of the prodrome, and thus of the beginning of the first psychotic episode, is less problematic (Beiser, Erickson, Fleming, & Iacono, 1993), notwithstanding the currently held belief (see Claridge, 1985, 1994; Johns & Van Os, 2001; Van Os, 2003) that psychotic symptoms, although also characterized by some qualitative change, occur on a continuum with normal experiences. According to Loebel et al. (1992) and Beiser et al. (1993), the prodromal period in schizophrenia lasts around 2 years on average, whereas Häfner and collaborators in their very detailed and innovative "Age, Beginning, and Course" (ABC) study of schizophrenia (e.g., Häfner & Maurer, 1996; Maurer & Häfner, 1997) report a mean duration of 5 years. In some cases, prodromes of even 11 years or more have been reported (Häfner et al., 1991; Møller & Husby, 2000). The initial prodrome should be distinguished from the so-called relapse prodrome, the period before psychotic recidivism in patients that had already suffered from a psychotic (and in this case schizophrenic) disease (e.g., Herz & Melville, 1980; Malla & Norman, 1994). Moreover, the initial prodrome must not be confused with Huber's (e.g., Huber, 1995) "Vorposten" or outpost syndrome, which may resemble the initial prodrome cross-sectionally, but which resolves spontaneously without progressing to psychosis. Because many prodromal symptoms, especially those involved in the beginning of the prepsychotic period, are ordinarily considered nonspecific or uncharacteristic, the concept of the initial prodrome is essentially a retrospective concept. This has prompted McGorry and Singh (1995) to speak of an "at-risk mental state," meaning that the clinical picture will not invariably be followed by the onset of psychotic symptoms. Moreover, the state character of the alternative concept emphasizes the point that the prodrome, according to Yung and McGorry (1996a), must be distinguished from trait risk or vulnerability factors (e.g., Nuechterlein & Dawson, 1984), such as longstanding schizotypal personality features or a positive family history of schizophrenia. However, it must also be stressed that a complete distinction is simply impossible, because many prodromal symptoms possess both state- and trait-like qualities, due to feedback mechanisms that temporarily lead to higher levels of vulnerability and subsequently to more severe prodromal symptoms (Klosterkötter, 1996; Nuechterlein & Dawson, 1984; Parnas, 1999). The relatively long duration of the initial prodrome also hampers a clear distinction between state and trait risk factors. Hence, for all practical purposes, the schizophrenic prodrome largely coincides with schizotypy or "psychosis-proneness" (see also, Walker & Gale, 1995).

Characteristic and Uncharacteristic Features

Strictly speaking, the concept of the schizophrenic prodrome implies that schizophrenia must be regarded as a psychotic disease. Indeed, this is precisely the reason why some prodromal features, such as social withdrawal and general decreased drive (see, e.g.,

Varsamis & Adamson, 1971), are termed uncharacteristic, whereas others, such as delusional thinking and perceptual disturbances (e.g., Chapman, 1966; Møller & Husby, 2000), which are often interpreted as attenuated psychotic symptoms, have been called relatively specific. However, in both Kraepelin's (1913) and Bleuler's (1911) original characterization of schizophrenia (or dementia praecox), the so-called "Grundsymptome" (fundamental symptoms) constitute the most defining and specific features of the disorder, whereas hallucinations and delusions (and also catatonic symptoms) are held to be only accessory and highly unspecific. In Bleuler's (1911) view, for instance, schizophrenia is characterized "by a specific type of alteration of thinking, feeling, and relation to the external world which appears nowhere else in this particular fashion" (p. 6). This alteration consists mainly of the four A's—disturbances of association and affectivity, autism, and ambivalence—but refers also to abnormalities in attention, volition, and sense of identity. A highly similar list of basic symptoms was provided by Kraepelin (1913, p. 936, 747), who defined them as "the invariable and permanent fundamental features of dementia praecox, accompanying the whole evolution of the disease" (Kraepelin, 1904, p. 26). However, two of Bleuler's "A" features—autism and ambivalence—were not regarded by Kraepelin to be core symptoms, because they were felt to be not always present in the end states of dementia praecox.

In contrast to the fundamental symptoms, the accessory symptoms were assumed to be not caused by the essential features of the disease process, but by circumstances which are only loosely associated with it (Kraepelin, 1913, p. 936). Indeed, a diagnosis of dementia praecox could be made in the absence of hallucinations and delusions, for these features, although frequently present, may develop "in very different degrees or be altogether absent, or disappear, without the fundamental features of the disease or its course and issue being in any way affected" (Kraepelin, 1904, p. 27). Hence, patients that showed only fundamental symptoms were diagnosed by Kraepelin and Bleuler as suffering from a subtype of dementia praecox or schizophrenia, viz., dementia or schizophrenia simplex. Although a psychotic definition of schizophrenia has been very much emphasized since the introduction of Schneider's (1950) list of first-rank symptoms (e.g., the hearing of one's own thoughts, and the hearing of voices commenting on one's actions), research has shown that these symptoms—which were later on even made an important component of, for instance, the *Research Diagnostic Criteria* (RDC; Spitzer, Endicott, & Robins, 1978), the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised* (DSM-III-R; American Psychiatric Association [APA], 1987), *The Tenth Revision of the International Classification of Diseases and Related Health Problems* (ICD-10; World Health Organization [WHO], 1992), and the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV; APA, 1994) criteria for a schizophrenic disorder—are far from pathognomonic (see, e.g., Peralta & Cuesta, 1999; Ross et al., 1990). In a recent article by Tsuang, Stone, and Faraone (2000), the possibility that psychotic symptoms have a separate status apart from the core symptoms of schizophrenia was also considered. Based, for instance, on a study by Bell, Dudgeon, McGorry, and Jackson (1998), it was concluded that psychosis is only "the 'fever' of severe mental illness—a serious but nonspecific indicator" (p. 1045). Although one may doubt the nonspecificity of every psychotic symptom, because hallucinations with complete sensory distinctness, in particular the idea of influence on thought and will and the delusion of physical, especially sexual influences were observed by Kraepelin (1913, pp. 958, 1193) as highly typical for dementia praecox and not for manic-depressive psychosis (see also, Kendler, 1986), Tsuang et al.'s (2000) assertion seems at least correct with respect to many psychotic symptoms. Furthermore, the fact that similarities exist between different psychotic illnesses does "not necessarily imply that the underlying disorders lie on the same continuum" (Tsuang

et al., 2000, p. 1045). In general, however, psychosis was seen by Tsuang et al. (2000) as “an end-state condition that in comparison with other indicators [which form part of what Tsuang et al. term the syndrome of schizotaxia], is more distal from schizophrenia’s causes and pathophysiology” (p. 1045). The correspondence with Kraepelin’s view on the etiology of accessory symptoms seems obvious.

Prodromal and Fundamental Symptoms

Despite the fact that the symptoms believed to characterize schizophrenia, and the diagnostic importance attributed to them, have varied since the beginning of the 20th century, the concept of the schizophrenic prodrome shows several similarities with the definition of Bleuler’s and Kraepelin’s Grundsymptome. This is especially true with respect to such prodromal symptoms as affective flattening, decline of interest, and social withdrawal, that are milder forms of negative symptoms of schizophrenia (see for the negative–positive distinction, e.g., Andreasen & Olsen, 1982; Crow, 1985). The latter symptoms have been interpreted by Andreasen (1982) and Tsuang, Gilbertson, and Faraone (1991) to be the equivalent of Bleuler’s and Kraepelin’s fundamental symptoms. However, there is also a certain similarity between at least some psychotic-like prodromal symptoms and some fundamental features listed by Kraepelin (1913), notably the group of disorders associated with the “loosening of the inner unity of the psychic life” (p. 936), manifesting itself in, for instance, “the disorders of association, described by Bleuler, incoherence in the train of thought, in the sharp change of moods as well as in desultoriness and derailments in practical work” (p. 747). It is reassuring to see that also in Tsuang et al.’s (2000) characterization of the cluster of schizotaxic phenomena assumed to be core features of schizophrenia, negative symptoms—together with psychosocial dysfunction, brain abnormalities, and neuropsychological impairments—play an all-important role, because these phenomena, if compared with positive features, appear to reflect more directly the genetic and early environmental bases of schizophrenia (see also below), and, hence, provide an accurate measure of susceptibility to schizophrenic breakdown (Tsuang, Stone, & Faraone, 2001). Although reminiscent of Kendler’s (1985) “familial” form of schizotypal personality disorder (SPD) or of, for instance, *DSM-III-R* SPD minus psychotic-like symptoms, Faraone, Green, Seidman, and Tsuang (2001) point out that these concepts are not considered to be identical with schizotaxia, because they observed that the number of nonpsychotic relatives of schizophrenics with negative symptoms (and/or neuropsychological deficits) exceeded the number of relatives diagnosed with familial SPD. Whatever the merits of the schizotaxia concept above Kendler’s familial form of SPD, from a prodromal perspective both negative and psychotic-like or positive features are important to give a full description of the schizophrenic prodrome. This is, for instance, apparent in the *DSM-III-R* (APA, 1987) listing of prodromal features, which is virtually identical to the list of negative and positive SPD criteria in *DSM-III-R*, and in Møller and Husby’s (2000) description of the experiential and behavioral core features of the schizophrenic prodrome (see also below). A further resemblance to the Grundsymptome is apparent with respect to the prepsychotic status of the initial prodrome, which fits exactly with Kraepelin’s “temporal” characterization of dementia simplex. “If one will,” writes Kraepelin (1913), “one may also regard dementia simplex in a certain way as the first period of dementia praecox. The cases which belong to it halt on one of the steps which form this period, while in the remaining forms there occurs progress of the malady beyond that point” (p. 766).

Although it transpires from both Kraepelin’s and Tsuang’s description of schizophrenia, that the concept of the schizophrenic prodrome, seen as a period of initial disturbance

which precedes the emergence of the characteristic psychotic symptoms, is actually a misnomer; it is still a period of prepsychotic disturbance in the context of the development of a schizophrenic psychosis. However, because in all studies about prepsychotic schizophrenic phenomena this period has been typically denoted the schizophrenic prodrome, we will continue to use this conceptually ill-devised term at least in this article. Likewise, we will continue to use the terms *(un)specific* and *(un)characteristic* in the sense attributed to them in the literature about precursors of schizophrenic psychosis. However, in the Discussion section we will return to this issue.

Symptoms and Symptom Categories

The main methodology to investigate the prodromal phase of schizophrenia has been the retrospective reconstruction of this phase by means of patient interviews after recovery from first-episode psychosis, and interviews with relatives and friends (Yung & McGorry, 1996a). Although problems of recall may hamper the validity of this method (but see, e.g., Cutting & Dunne, 1989), these problems may be overcome by the use of specific instruments such as the Interview for the Retrospective Assessment of the Onset of Schizophrenia (IRAOS; Häfner et al., 1992). Furthermore, Häfner et al. (1992) consider retrospective studies based on large first admission samples to be the only practical way to collect sufficient data on the initial prodrome and the emergence of psychotic symptoms in schizophrenia.

Several symptoms have been noted to form part of the schizophrenic prodrome. For instance, in Varsamis and Adamson's (1971) retrospective study five symptom categories could be distinguished—e.g., “disturbances of affect,” “disturbances of drive,” and “changes in the relation to the environment”—that included such features as anxiety, suspiciousness, slowed thinking, and schizoid withdrawal. Another example is Møller and Husby's (2000) investigation in which eight groups of prodromal experience were discerned—e.g., “disturbance of perception of self,” “neuroticlike disturbances,” and “attenuated delusional ideas or perceptions”—as well as four dimensions of prodromal behavior, e.g., “shifts of interest,” and “withdrawal and extreme social avoidance.”

To survey the range of subjective and observable symptoms that are mentioned in the clinical literature, Yung and McGorry (1996a) present a comprehensive list of prodromal phenomena that is based on 24 separate studies published between 1911 and 1994 (e.g., Bowers, 1968; Cameron, 1938; Conrad, 1958; Hambrecht, Häfner, & Löffler, 1994; Huber, Gross, Schuttler, & Linz, 1980). In this summary, 33 prodromal features could be distinguished that were classed under 7 categories: (a) “neurotic” symptoms (anxiety, restlessness, anger/irritability), (b) mood-related symptoms (depression, anhedonia, guilt, suicidal ideas, mood swings), (c) changes in volition (apathy/loss of drive, boredom/loss of interest, fatigue/loss of energy), (d) cognitive changes (disturbance of attention/inability to concentrate, preoccupation/daydreaming, thought blocking, reduced abstraction), (e) physical symptoms (somatic complaints, loss of weight, poor appetite, sleep disturbance), (f) other symptoms (obsessive compulsive phenomena, dissociative phenomena, increased interpersonal sensitivity, change in sense of self/others/the world, change in motility, speech abnormalities, perceptual abnormalities, suspiciousness, change in affect), and (g) behavioral changes (deterioration in school/work/role functioning, social withdrawal, impulsivity, odd behavior, aggressive/disruptive behavior).

Patterns of Prodromal Change

Besides the symptoms themselves, several studies have addressed the patterns of change in the schizophrenic prodrome. According to Yung and McGorry (1996a), previous authors

have generally taken two different viewpoints regarding the sequence of changes that leads to psychosis. In the first of them, the assumption is held that the schizophrenic prodrome starts with nonspecific and seemingly neurotic features, and that this phase is followed by the emergence of relatively specific psychotic-like symptoms heralding impending psychosis (see, e.g., Cameron, 1938; Docherty et al., 1978; Gross & Huber, 1989; Meares, 1959). The second hypothesis, which is virtually only represented by Chapman (1966; McGhie & Chapman, 1961), assumes the reverse order, in that attenuated psychotic symptoms (e.g., disturbances of attention, perception, and thinking) are believed to come first, later on followed by both psychotic phenomena and reactive "neurotic" symptoms.

Although the issue has not been completely decided, the first hypothesis seems at least correct in broad outline. The most important investigation in this respect has been conducted by Häfner et al. (1995) in the already cited ABC study of schizophrenia (see also Maurer & Häfner, 1997). Using the IRAOS in a sample of 232 broadly defined International Classification of Diseases (9th revision; ICD-9; WHO, 1978) schizophrenic patients with a first psychotic episode, it could not only be demonstrated that among the 10 most frequently cited earliest symptoms (e.g., restlessness, poor concentration, anxiety, and lack of energy) no positive features appear, but also that in nearly 75% of the cases the first admission to hospital is preceded by two prephases. The prepsychotic or prodromal period is characterized by unspecific and negative symptoms (depressed mood, social withdrawal, disturbance of affect, etc.). The psychotic prephase ends when the number of clearly psychotic symptoms (e.g., delusions of persecution, auditory hallucinations, and thought block) is at its maximum; it is sometimes followed by a short latency period prior to first admission. Actually, the two prephases were distilled from the observation that the accumulation of positive, negative, and nonspecific symptoms each year observed over a period of 15 years before hospital admission suggested an almost parallel, continuous, and exponential rise with a time lag of, on average, 5 years for the positive symptoms (see also Häfner & Maurer, 1991). That no psychotic-like features (such as delusional mood and depersonalization) are mentioned in the description of the schizophrenic prodrome is due to the fact that these symptoms, although listed in the IRAOS (see Häfner et al., 1992), were not studied in the present investigation. Only in 6.5% of the cases was schizophrenia found to start with positive symptoms only, whereas in about 20% the onset was characterized by a mix of positive and negative and/or uncharacteristic features. The usual delay in the development of positive symptoms is also apparent in a retrospective study by Yung and McGorry (1996b). Interviews with 21 first-episode patients after recovery from psychosis brought several prodromal features to light, with symptoms being a mixture of nonspecific phenomena—including, for instance, anxiety, irritability, low energy, and social withdrawal—and attenuated psychotic symptoms, such as perceptual disturbances and delusional mood. Concerning the course of the schizophrenic prodrome, the uncharacteristic features tended to occur early, whereas the psychotic-like features occurred much closer to the onset of the subsequent psychotic episode.

Relationship Between Positive and Negative Symptoms

One further observation in the research carried out by Häfner and collaborators has to do with the kind of relationship between negative and positive symptom categories or dimensions. Although it has been often reported in the literature (e.g., Crow, 1985; Lewine, Fogg, & Melzer, 1983) that both symptom groupings are independent, there are several

methodological reasons to distrust this conclusion (Czobor & Volavka, 1996; Maurer & Häfner, 1991). Moreover, the above-mentioned finding of an almost parallel development of negative and (delayed) positive symptoms leads to the expectation that both syndromes must have something in common. Indeed, nearly all correlations between scores for negative and positive symptoms, calculated for each year in the period of 15 years prior to hospital admission, for each month in the final pre-admission year, and for several time-points in a period up to 3 years after admission, were reported by Häfner et al. (1995) to depart significantly and positively from zero, with values varying between 0.15 and 0.45 approximately. Although Häfner & Maurer (1991) interpret these correlations and the almost parallel course in terms of a common etiological factor, attention must be drawn to the fact that among biological relatives of schizophrenics predominantly negative symptoms have been reported (see, e.g., Grove et al., 1991; Gunderson, Siever, & Spaulding, 1983; Kendler, McQuire, Gruenberg, & Walsh, 1995; Tsuang et al., 1991). This suggests that negative symptoms, rather than positive ones (and notwithstanding their statistical interdependence), are a more direct expression of the biological-genetic basis of schizophrenia. Research findings of Battaglia, Bernardeschi, Franchini, Bellodi, and Smeraldi (1995) which indicate that many patients with "clinical" SPD (Kendler, 1985) do not carry the genetic predisposition to schizophrenia are also of importance here, because Thaker, Moran, Adami, and Cassady (1993) could demonstrate that patients with "clinical" SPD show higher levels on the Chapman scales of perceptual aberration and magical ideation (see, e.g., Chapman, Chapman, & Miller, 1982) than patients with "familial" SPD. In addition, taking into account Kraepelin's (1913) and Tsuang et al.'s (2000) assertion (see above) that psychotic symptoms in schizophrenia do not relate strongly to schizophrenia's underlying disease process or to its causes, we may, perhaps, conclude that the genetic predisposition to schizophrenia may manifest itself first in negative symptoms, but that these "familial" prodromal symptoms in turn seem to constitute a fertile soil for the development of psychotic-like and later on, psychotic symptoms. The latter symptoms, therefore, may reflect the genetic predisposition only relatively weakly or in an indirect manner.

Finer Distinctions and Stages of Onset

Besides the distinction between negative and positive prodromal symptoms, some further distinctions have been made. Even without focusing on attenuated psychotic symptoms, Maurer and Häfner (1997) mention five putatively subsequent stages in the prepsychotic prodromal period as observed in the sample of 232 first-episode patients mentioned above: Stage 1—a depressive stage; Stage 2—a dysphoric stage, Stage 3—energy loss, Stage 4—a second dysphoric stage, and Stage 5—a stage of transition prior to the emergence of the first psychotic symptom. However, as the symptoms in these stages are grouped together on the basis of their *average* time differences from first symptom onset until first hospitalization, no allowance is made for the widely varying duration of the schizophrenic prodrome (see Møller & Husby, 2000, for a summary of duration data). To illustrate this issue, note that some of the symptoms found by Häfner et al. (1995) to initiate the prodrome relatively often (e.g., restlessness, poor concentration, and suspicion; see above) are now classed under the Stages 3 and 4.

In contrast to this, a naturally occurring sequence of stages (with unspecific and negative features preceding positive symptoms) is presented in Docherty et al.'s (1978) study. Reviewing the literature on the process of psychotic decompensation in schizophrenia (e.g., Arieti, 1955; Cameron, 1938; Chapman, 1966; Conrad, 1958; Sullivan, 1962; Varsamis & Adamson, 1971), the authors claimed to have noted a remarkable

concordant description not only of identifiable premonitory features but also of a regular and sequential unfolding of psychological states before and during psychotic breakdown. Given this uniformity, it was felt to be relatively simple to construct a comprehensive picture of the decompensation process in which five stages of onset were discerned:

1. Overextension. "During this phase the person begins to experience a sense of being overwhelmed. This seems secondary to either external demands or unrelenting conflict" (Docherty et al., 1978, p. 426). The authors mention several features and expressions to characterize this stage, such as "nervousness," "overstimulation," "increasing anxiety," "feeling of losing one's grip," and "neurotic process." However, disregarding Chapman's (1966) study because it does not describe the typical sequence of changes that leads to psychosis (see above), "overstimulation"—that according to Chapman initiates schizophrenia—then must be skipped from this list, as well as the overall designation "overextension." A more appropriate term for this stage would be *Fear* or *Anxiety*.
2. Restricted consciousness. "During this phase a variety of mental phenomena appear that seem to bring about a limitation of the person's range of thought" (Docherty et al., 1978, p. 426). Characteristic features include withdrawal, isolation, emotional blunting, narrowed attention, boredom, and feelings of alienation.
3. Disinhibition. "During this phase relatively unmodulated impulse expression appears. This period may bear a close resemblance to hypomania" (Docherty et al., 1978, p. 426). Perhaps following a condition in which the patient experiences an insoluble impasse, this stage is characterized by rage, upsetting ideas, escape of dangerous impulses, rapid oscillation in mood, efforts to relieve boredom, and feelings of creative release. Again disregarding Chapman's (1966) study, perceptual alteration is not included.
4. Psychotic disorganization. Three subphases are recognized: (a) destructuring of the external world (with increasing perceptual and cognitive disorganization), (b) destructuring of self (with loss of self-identity), and (c) total fragmentation (with complete loss of self and control). Some listed features include tendency to misinterpretation, concretization, destructuring of perception, ideas of reference, and complete disorganization.
5. Psychotic resolution. This stage is marked by less anxiety and psychotic reorganization, either of a delusional kind (schizophrenia, paranoid type) or involving massive denial of all unpleasant affect and responsibility (hebephrenic type).

Cause-Effect Relationships

With regard to the processes involved in the sequential unfolding of the five onset stages, nothing can be said with certainty. However, some of the features described by Docherty et al. (1978) as present in a certain phase may be considered the direct *cause* of other features occurring in the same or in a subsequent phase. This becomes particularly apparent when relating Stages 2, 4, and 5 to each other by drawing attention to Bleuler's (1911) concept of autism, which he defined as "the predilection for fantasy as against reality and the inclination to divorce oneself from reality" (p. 10). Not only are the origins of the destructuring of the outer world and of the self (Stage 4) located in the detachment from reality as described by Bleuler, but the predominance of the inner life is likely to be causally dependent on the presence of withdrawal and isolation (Stage 2). Indeed, as stated by Millon and Davis (1996) in their explanation of the development of schizotypal symptoms:

The more individuals turn inward, the more they lose contact with the styles of behavior and thought of those around them. As they become progressively estranged from their social environment [also a Stage 2 phenomenon; see Docherty et al.'s (1978) feelings of alienation], they lose touch with the conventions of reality and with the checks against irrational thought and behavior that are provided by reciprocal relationships (p. 613).

A similar view was expressed by Allen, Coyne, and Console (1997) who speak about a process of inward flight by which a state of intense absorption—a variable strongly related to fantasy proneness (see, e.g., Merckelbach, Horselenberg, & Muris, 2001)—leads to a change in the sense of self (depersonalization) and reality (derealization), with the effect that these features render individuals vulnerable to psychotic experience (see also Allen & Coyne, 1995). Within the psychotic realm, delusions (Stages 4 and 5) are sometimes interpreted to consist of rational attempts to explain unusual perceptual events (Stage 4; Chapman, 1966; Maher, 1988; Maher & Ross, 1984; Roberts, 1992). Accordingly, of the prodromal symptoms, psychotic-like perceptual disturbances may be expected to causally precede delusional thinking. Also pointing to Kretschmer's (1942) observation that schizoid withdrawal is at first associated with oversensitivity (e.g., "mimosa-like" behavior, shyness, and nervousness), withdrawal and isolation (Stage 2) are the likely causal consequences of Stage 1 phenomena, in this case fear or social anxiety in particular.

One further aspect that is of importance here concerns Kretschmer's (1942) observation of a special relationship between oversensitive and insensitive features. Both components were not only observed to be usually present at the same time, albeit in quite different relative proportions, but this mixture (called the *psyschaesthetic proportion*; Kretschmer, 1942, p. 163) was also found to show a transition in the course of time from the hyperesthetic to the anesthetic pole in most cases. Although the exact mechanisms responsible for this change are not clearly indicated, it transpires from Kretschmer's writings (see Kretschmer, 1942, p. 165) that autistic withdrawal initially has the function to dampen all painful feelings associated with hypersensitivity, and, hence, withdrawal seems to play a primary role in the transition of the feeling tone in the direction of anesthesia. Even apart from this mechanism, a general emotional cooling is expected to result from social isolation. According to Kretschmer (p. 170), three clusters of anesthetic features may be discerned: a cluster centering around, respectively, emotional dullness or a passive lack of feeling; emotional coldness or an "active unfeelingness of all kinds" (p. 176) (e.g., rudeness, egoism, lack of consideration, cruelty, mordacity, and active hostility); and, finally, emotional indifference, a variant that is partly active and partly passive. At least the Stage 2 qualifications of rage and escape of dangerous impulses in Docherty et al.'s (1978) scheme bear a certain similarity to the anesthetic features described by Kretschmer as emotional coldness. Hence, a causal relationship can be inferred between social withdrawal and emotional blunting—the passive type of anesthesia—(both Stage 2 phenomena), as well as between withdrawal (Stage 2) and active hostility (Stage 3) or other emotionally cold features. Selecting hostility as the main symptom here, this latter relationship might be mediated by one of the other qualities listed by Kretschmer, viz., egoism. Furthermore, a causal relationship can be speculated on between emotional blunting, on the one hand, and depersonalization and derealization on the other, because it seems plausible to regard emotional blunting as the first manifestation of progressive estrangement.

With respect to the causal chains between Stages 1, 2, 3, 4, and 5, several studies searching for the most heritable components of schizotypal personality disorder in its relation to schizophrenia (e.g., Brunke, Pogue-Geile, Garrett, & Hall, 1991; Ingraham, 1993; Kendler, Gruenberg, & Strauss, 1982; Torgersen, Onstad, Skre, Edvardsen, &

Kringlen, 1993; Torgersen et al., 2002) converge on at least the relative importance of seclusive/withdrawn behavior and excessive social anxiety. Hence, extending an earlier conclusion, we may perhaps state that the genetic predisposition of schizophrenia expresses itself first of all in social anxiety and isolation (Stages 1 and 2), then in other negative symptoms (Stage 2) to a somewhat lesser degree, and finally, but the least strongly, in active hostility (Stage 3) and in psychotic-like and psychotic features (Stages 4 and 5).

Extension of the Network

Besides the symptoms derived from the writings of Bleuler, Kretschmer, and others, there are three other features that extend the network of causal relationships just indicated: suspiciousness, apathy, and cognitive derailment. Suspiciousness could be just another symptom mediating between social withdrawal and rage. This expectation is grounded in the observation that the suspiciousness scale of the Dimensional Assessment of Personality Pathology-Basic Questionnaire (DAPP-BQ; see, e.g., Livesley & Jang, 2000) was found to load on two almost uncorrelated factors: one "Emotional Dysregulation," relating among other variables to social avoidance, and another "Dissocial," associated with such hostility-related features as rejection and callousness (Livesley, Jang, & Vernon, 1998; Van Kampen, 2002). Apathy was included in the network as a symptom causally dependent on depersonalization and derealization, because it was felt that these changes in particular could lead to such confusion, perplexity, and ambivalence that the occurrence of any form of goal-directed activity will be seriously hampered. Finally, cognitive derailment was added to the symptoms already cited, because it may bridge the cleft (see Millon & Davis, 1996) between progressive estrangement (depersonalization and derealization) and psychotic irrational thought. This feature was introduced as a combination of Andreasen's concept of "alogia" (see, e.g., Andreasen, 1984) and *subclinical* or attenuated formal thought disorder, emphasizing both vagueness in thinking, the loosening of associations, and the tendency to use inappropriate words. Actually, this combination was based on the assumption that the origins of incoherence and tangentiality lie in vague or impoverished thinking.

Representation of the "Full" Schizophrenic Prodrome

It is very important to note that the symptoms involved in the above-mentioned causal statements can be easily located in nearly all symptom categories distinguished by Yung and McGorry (1996a) based on their survey of prodromal features (see above). Fantasy proneness, for instance, refers to the same phenomenon as preoccupation/daydreaming, which is classed by Yung and McGorry under the heading "cognitive changes." Likewise, social withdrawal and apathy are classed under "behavioral changes" and "changes in volition," respectively. Other examples are (a) depersonalization and derealization which correspond to dissociative phenomena and change in sense of self/others/the world rubricated under "other symptoms," (b) cognitive derailment which shows some similarity with thought blocking and speech abnormalities, listed under, respectively, "cognitive changes" and "other symptoms," and (c) hostility which corresponds to anger/irritability and aggressive/disruptive behavior, categorized as "neurotic symptoms" and "behavioral changes," respectively. The only exception concerns Yung and McGorry's category of physical symptoms, but these symptoms seem secondary rather than fundamental. Hence, assuming that the above-mentioned causal pathways can be shown to be valid, the network offers a representative summary of the schizophrenic prodrome in all its facets. It follows that a dependable, meaningful, and conceptually satisfying definition of the

schizophrenic prodrome can be made, including its temporal unfolding in the direction of psychosis, by emphasizing the symptoms listed above and the causal network in which these symptoms are embedded.

The Schizotypic Syndrome Questionnaire: Symptoms and Causal Pathways

The Schizotypic Syndrome Questionnaire (SSQ) was developed to measure the schizophrenic prodrome as defined above. The inventory is comprised of separate scales for 12 symptoms: social anxiety (SAN), active isolation (AIS), living in a fantasy world (FTW), affective flattening (AFF), egocentrism (EGC), hostility (HOS), feelings of alienation (ALN), perceptual disturbances (PER), delusional thinking (DET), suspicion (SUS), apathy (APA), and cognitive derailment (CDR). The first nine scales refer to those prodromal features that seem to be involved in the causal relationships noted in the writings of Bleuler (1911), Kretschmer (1942), and Allen, Coyne, and Console (1997). The three remaining scales (SUS, APA, and CDR) are the scales for the features that are hypothesized to amplify the network derived from that literature. Each of the 12 scales consists of 9 items in the form of statements about behavior and feelings, amounting to a total of 108 items. The items are answered on a 4-point scale by putting a circle around *YES*, *yes*, *no*, or *NO*, indicating the degree to which each statement applies to the subject. To illustrate the contents of the SSQ scales, Table 1 lists for each scale the first, fifth, and ninth item, respectively.

Three samples were used for the construction of the SSQ. These samples consisted of, respectively, 381, 265, and 329 normal subjects drawn from the patient files of 11 general practitioners from the Dutch towns Apeldoorn and Breda (Sample AB), two general practitioners from Leiden (Sample L), and five from Haarlem (Sample H). Nine of the 12 SSQ scales were developed in Sample AB (SAN, AIS, FTW, AFF, ALN, PER, DET, SUS and CDR), two scales (EGC and HOS) in L, and one (APA) in H. The PER and DET scales are essentially short versions of Chapman, Edell, and Chapman's (1980) Perceptual Aberration Scale and of Eckblad & Chapman's (1983) Magical Ideation Scale, although two new items were included in the DET scale. For the construction of the SSQ scales, only those items were selected for which it could be demonstrated that the corrected item-total correlations for the items belonging to a scale were always higher than the correlations of the same items with each of the remaining scales. This was done to safeguard the conceptual clarity of each scale as much as possible. Data about the internal consistency, factor structure, and validity of the 12 SSQ scales, as found in the present investigation, will be reported elsewhere (see, e.g., Van Kampen, 2005; Van Kampen & Deijen, 2005). However, the results can be summarized by stating (a) that the Cronbach alpha coefficients for 8 SSQ scales in the total HGA sample (see below) turned out to vary between 0.85 and 0.91, and for 4 SSQ scales (PER, DET, EGC, and HOS) between 0.77 and 0.79; (b) that a principal components analysis of the 12 SSQ scales has led to the extraction of three (correlated) factors—interpreted as Negative Schizotypy, Positive Schizotypy, and Asocial Schizotypy—that are similar to the well-known schizotypy dimensions reported in the literature (e.g., Claridge et al., 1996; Vollema & Van den Bosch, 1995) Anhedonia and Cognitive Disturbances (these two factors combined), Unusual Experiences, and Impulsive Nonconformity; (c) that the SSQ scales were found to correlate as expected with the scales and factors of Raine's (1991) Schizotypal Personality Questionnaire, the Dissociative Experiences Scale-II (Carlson & Putnam, 1993), the Creative Experiences Questionnaire (Merckelbach et al., 2001), and Van Kampen's (1997) 4-Dimensional Personality Test (4DPT) and its successor, the 5DPT (Van Kampen, 2005);

Table 1
Examples of SSQ Items

Scale		
SAN	Social Anxiety	I often feel frightened when somebody asks me something unexpectedly. I usually have great difficulty in adapting to other people. Actually, I am afraid of people.
AIS	Active Isolation	I do not want to have anything to do with other people. I prefer to keep my distance from others. I prefer to avoid people.
CDR	Cognitive Derailment	I often find that other people have difficulty understanding my words. Sometimes I am unable to make sense of my own words. I notice that I am sometimes not fully aware of what I am talking about.
AFF	Affective Flattening	I tend not to experience strong emotions. I appear to have lost the ability to have any feeling. I am at times unable to feel anything.
PER	Perceptual Disturbances	Occasionally parts of my body seem to be dead or unreal. Ordinary colors sometimes seem too bright (without me taking drugs). Over a stretch of several days, I sometimes have such a heightened awareness of sights and sounds that I cannot shut them out.
FTW	Fantasy World	I can create a completely private world in my own thoughts. In my fantasies I tie all sort of things together as it pleases me. I am sometimes so engaged in my daydreams that I experience reality as disrupting.
SUS	Suspicion	I tend to be suspicious of other people. I am rather distrustful of most people. I sometimes wonder whether people mean what they say.
APA	Apathy	I often neglect my work. Sometimes days pass without me really doing anything. Every effort puts too great a strain upon me.
ALN	Alienation	It sometimes seems as if I am a prisoner in my own world. It sometimes seems as if there is a wall between me and my surroundings. I sometimes feel alienated from myself.
DET	Delusional Thinking	Occasionally I have the silly feeling that a TV or radio broadcaster knew that I was listening to him or her. Occasionally I have the feeling that certain thoughts of mine really belong to someone else. I think you can win in a game of chance by concentrating beforehand on its outcome.
EGC	Egocentrism	It seems quite natural to me that people should serve me and satisfy my needs. Actually I am only interested in myself. I usually follow only my own desires in what I do.
HOS	Hostility	I often dislike others. I sometimes behave very hostile towards people. I find it difficult to forgive other people.

and (d) that in a group of subjects that were instructed to follow a horizontally moving target under three conditions of speed (low, medium, and high), the subgroup scoring high on the general factor of the SSQ showed significant impairments in the low- and high-speed conditions in global SPEM or smooth pursuit eye movement dysfunction, which is one of the most dependable biological markers of the genetic liability for schizophrenia (Van Kampen & Deijen, 2005).

The causal network supposed to exist between the 12 prodromal features measured by the SSQ is depicted in Figure 1. As can be seen from the figure, the ordering of the

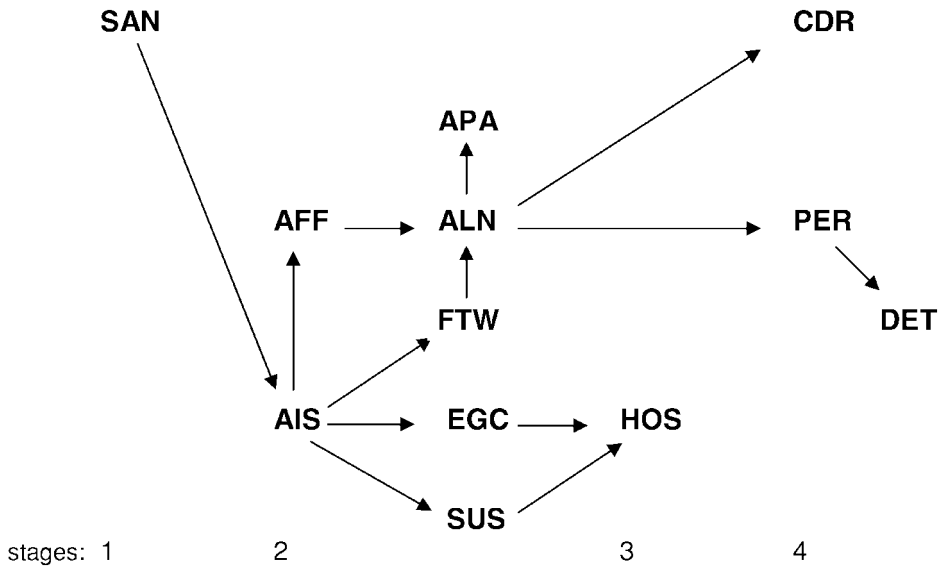


Figure 1. A proposed network of causal relationships. SAN = social anxiety; AIS = active isolation; AFF = affective flattening; APA = apathy; ALN = alienation; FTW = fantasy world; EGC = egocentrism; SUS = suspicion; HOS = hostility; CDR = cognitive derailment; PER = perceptual disturbances; DET = delusional thinking. The numbers 1, 2, 3, and 4 refer to the first four stages in the scheme of Docherty et al. (1978).

symptoms is also determined, admittedly rather roughly, by their place in Docherty et al.'s (1978) scheme of schizophrenic onset stages. However, the position for three symptoms had to be derived from the localization of other features or was estimated based on the information about the DAPP-BQ (see above). For instance, living in a fantasy world (FTW) was considered a Stage 2 phenomenon, because the same was held to be the case by Docherty et al. for the subsequently manifesting feelings of alienation (ALN). The place of suspicion (SUS) in the model was dictated by the finding that the Suspiciousness scale of the DAPP-BQ correlated slightly higher with the DAPP-BQ Social Avoidance scale ($r = 0.59$) than with the hostility-related features rejection and callousness (mean $r = 0.42$) (Van Kampen, 2002), so this feature was localized in Stage 2. Likewise, EGC or egocentrism was put in Stage 2, because of the finding (Van Kampen, 2002) that the DAPP-BQ Narcissism scale—with its emphasis on egocentrism—resulted in a similar (but less convincing) pattern of r values; the correlation with social avoidance being $r = 0.47$, and the mean correlation with the two hostility-related scales $r = 0.43$.

Model Testing and Model Building

One of the principal aims of this study is to test the model of unfolding portrayed in Figure 1 by means of structural equation modeling, applying LISREL-8, in a relatively large sample. A priori, it is unlikely that this causal network model would immediately result in an adequate fit; the testing actually has the purpose of building a model, starting, of course, with the network depicted in Figure 1. Although some preliminary LISREL analyses have already been carried out in sample H, this sample was considered too small to give dependable results. Moreover, in this sample only nine scales of the SSQ were used for the LISREL testing. Therefore, a much larger group of 771 normal subjects was formed, extending the original H sample ($n = 329$) by two new samples: G ($n = 228$) and

A ($n = 214$). The insertion of H (instead of AB or L) in the total HGA sample was dictated by the fact that only in this subsample the scores on all 12 SSQ scales were known.

Use of a Normal Sample

Although it is clear that the prodromal features measured by the SSQ can be found in high-risk samples that are defined on the basis of the presence of premorbid vulnerability markers—for instance, a positive family history for schizophrenia, smooth pursuit eye movement dysfunctions, and certain behavioral abnormalities in childhood (see, e.g., Davidson et al., 1999; Levy, Holzman, Matthysse, & Mendell, 1993; Mednick & Olin, 1996; Walker & Lewine, 1989), a general population sample was used for several reasons.

First, following a high-risk strategy seems very time-consuming and impractical because of the need for a relatively large sample. Second, the testing of the SSQ model does not so much depend on the height of the SSQ scores as on their mutual relationships. If it is assumed that the correlations between the various SSQ scales demonstrate a similar pattern for both relatively high- and relatively low-scoring subjects, the use of a normal sample seems plainly justified. Third, the continuum hypothesis, to which this assumption implicitly refers, seems well supported. In several studies discussed by Johns and Van Os (2002), it was demonstrated that both hallucinatory and delusion-like experiences are present in considerable proportions in normal samples. In a study by Barrett and Etheridge (1992), for instance, between 30 and 40% of a college student sample reported the experience of hearing voices, and nearly half of the subjects indicated that the experience occurred at least once a month. Similar results were obtained in a study by Peters, Joseph, and Garety (1999) in which it was demonstrated that the score distributions of a normal and a psychotic sample on an inventory measuring a wide range of delusional beliefs did overlap each other substantially, with nearly 10% of the healthy sample even scoring above the mean of the psychotic group. Of particular interest is the finding (see Verdoux et al., 1998) that the typical co-occurrence of hallucinations and delusions in psychotic patients can also be observed in nonclinical samples for similar, but attenuated experiences. Indeed, psychotic-like, negative and other traits of psychosis-proneness or schizotypy, such as aberrant perceptions, social anhedonia, and paranoid ideation, have not only been studied meaningfully within nonclinical samples (see, e.g., Bentall, Claridge, & Slade, 1989; Claridge et al., 1996; Kendler & Hewitt, 1992; Muntaner, Garcia-Sevilla, Fernandez, & Torrubia, 1988; Venables, Wilkins, Mitchell, Raine, & Bailes, 1990), but these features have also been observed to usually give rise to the extraction of three or four factors (see, e.g. Vollema & Van den Bosch, 1995) that are strikingly similar to the dimensions found in factor analytic studies of schizophrenic symptoms (e.g., Arndt, Alliger, & Andreasen, 1991; Bilder, Mukherjee, Rieder, & Pandurangi, 1985; Liddle, 1987; Lindenmayer, Bernstein-Hyman, & Grochowski, 1994; Peralta, De Leon, & Cuesta, 1992). Although some form of discontinuity, which shows itself in a threshold effect, might also exist (see, e.g., Claridge, 1994; Van Os, 2003; Van Os, Verdoux, Bijl, & Ravelli, 1999), this “continuum-related discontinuity” effect has not been demonstrated convincingly. Thus, Eysenck’s conceptualization of a normality–psychosis continuum without any qualitative distinction (Eysenck, 1992) may also be adhered to as well as his more general view that psychiatric diagnostic categories do only represent points in a multidimensional space generated by the various personality factors postulated in his PEN model (Verma & Eysenck, 1973).

Whatever the possibility of an additional threshold effect, the above-mentioned findings do strongly suggest the existence of an unbroken continuum (or rather a set of

continua) between schizophrenia (including its psychotic manifestations) and normality. Such a model is also in line with Bleuler's (1911) recognition of the existence of a mild and relatively common form of schizophrenia, called *latent schizophrenia*, which exhibits its symptoms within normal limits. A similar view has also been expressed by Kretschmer (1942) by postulating a continuum ranging from schizophrenia to manic-depressive psychosis, via the less deviant groups of schizoid and cycloid personalities, and, finally, in the middle range of the continuum, via normal variants of what Kretschmer has called the *schizothymic* and *cyclothymic temperaments*.

Given the evidence and observations mentioned above (and notwithstanding the fact that some authors still believe that the categorical view on psychotic symptoms has some validity; see, e.g., Mullen, 2003), the continuum hypothesis with respect to both psychotic and nonpsychotic symptoms of schizophrenia appears to be valid, and, hence, the pattern of correlations between the characteristics measured by the SSQ is probably the same in schizophrenic psychotic patients, patients with SPD, subjects manifesting prodromal symptoms, and nonclinical subjects. Hence, the application of LISREL to analyze the covariance matrix obtained for the 12 SSQ scales in a general population sample is wholly appropriate.

Method

Subjects and Procedure

As already indicated, the total sample for the present investigation consists of 771 normal subjects. These subjects were drawn from the patient files of 5 general practitioners in Haarlem (H: 329 subjects), 4 in Gouda (G: 228 subjects), and 5 in Amsterdam (A: 214 subjects). Originally, 2,996 subjects, most often between 20 and 59 years of age (but in two A subsamples between 20 and 49, and 16 and 56 years, respectively) were approached by letter by their own family doctor with the request to fill in the SSQ. This questionnaire was usually sent to them in addition to the 4DPT or 4-Dimensional Personality Test (Van Kampen, 1997, 2000), but in a small subsample this was done together with Raine's (1991) Schizotypal Personality Questionnaire or SPQ in a Dutch translation by Vollema (see, e.g., Vollema & Hoijtink, 2000). The 4DPT and SPQ were administered to obtain some information about the validity of the SSQ scales; these and other validity findings will be reported in separate publications.

Because the patient files used for the present investigation were not completely brought up to date, only 2,893 subjects actually received a request from their family doctor to fill in the SSQ; hence the number of 771 subjects who filled in that instrument amounts to a response percentage of 26.7. Similar percentages were found in the subsamples H (28.1%), G (23.8%), and A (28.0%). Although these response percentages appear to be relatively low, the means and standard deviations obtained for the 4DPT scales in the present investigation were found to be almost identical to the means and standard deviations obtained in the original 4DPT standardization sample (Van Kampen, 1997). However, this latter sample of 626 normal subjects that was approached in a similar manner was associated with a much higher response percentage of 49.6. Hence, the present response rate does not appear to indicate an unusual or unrepresentative sample.

The group of 771 subjects consisted of 457 females, 280 males, and 34 subjects of unknown sex (and age). The mean age in this sample turned out to be 36.67 years with a standard deviation of 10.32. For the LISREL analyses conducted, the total group was randomly split in two subsamples, R1 and R2. These subsamples (without considering the 34 subjects of unknown sex and age) consisted of 375 and 362 subjects with a mean age and standard deviation of 36.95 ± 10.41 and 36.37 ± 10.23 years, respectively.

Statistical Analyses

The main reason for administering the SSQ was to build a model about the temporal unfolding of the schizophrenic prodrome, starting with the network of causal relationships depicted in Figure 1. Below, results will be presented that have been obtained after testing this network, including its modifications, by means of structural equation modeling, applying LISREL-8 (Jöreskog & Sörbom, 1995) to the covariance matrix derived from the scores on the 12 SSQ scales. Sample R1 was used for the construction of the model, whereas R2 was used in an attempt to replicate the finally selected model in R1.

In the LISREL testing as carried out both in R1 and R2 and in the total sample, the SSQ scales SAN, AIS, CDR, AFF, etc., refer to latent variables (ETA), each one indicated by one SSQ scale (y). Using Cronbach's alpha and the variances of y as calculated in the total sample, the regressions of y on ETA (LY) and the variances of the measurement errors in y (TE) were estimated. This was done because the alpha reliabilities of the PER, DET, EGC, and HOS scales were somewhat lower than the values found for the remaining scales (see above). The same LY and TE estimates were fed into all LISREL analyses. The parameters in BETA (path coefficients) not belonging to the model to be tested were fixed at zero. Of the PSI matrix, only the diagonal elements were left free. As it is known for large samples that the χ^2 statistic cannot reliably indicate a good fit (see, e.g., Marsh, Balla, & McDonald, 1988), three other indices were selected: the Root Mean Square Error of Attribution (RMSEA), the Comparative Fit Index (CFI), and the Standardized Root Mean Square Residual (SRMR), all three provided by the LISREL program. According to Hu and Bentler (1999), a satisfying fit is indicated by a CFI of at least 0.96, a RMSEA lower than 0.06, and a SRMR lower than 0.08. Furthermore, two joint criteria are specified by Hu and Bentler (1999), namely that a model should be retained if $CFI \geq 0.96$ and $SRMR \leq 0.10$ or the $SRMR \leq 0.10$ and $RMSEA < 0.06$. Of course, whether the path coefficients are significantly different from zero was also investigated. Besides one-sample analyses, a two-sample analysis using R1 and R2 was conducted, testing the invariance of the BETA matrix. To improve the original model in R1, both the modification indices (MI) and the expected change values in BETA were inspected, but, of course, the suggestions associated with these indices and values were not effected unless the additional pathways seemed likely to reflect truly causative influences.

Results

The LISREL testing of the originally postulated model portrayed in Figure 1 and of six models subsequently developed to give a better fit to the observed data was done in sample R1, using 371 of the 375 subjects. Table 2 (Models 1–7) presents the main findings by both specifying the pathways additionally selected for the various models and the goodness of fit indices associated with these models.

From Table 2, it can be seen that the originally postulated model already has a mediocre fit. However, there were several possibilities for improving the fit, finally leading to Model 7 in R1, which was considered the definitive model (at least in R1). For this model, the CFI and SRMR values indicate a satisfying fit, but the RSMEA value, at slightly above 0.06, indicates only a marginal ability of the model to reproduce the data. However, in terms of Hu and Bentler's (1999) criterion of a $CFI \geq 0.96$ in combination with a $SRMR \leq 0.10$, the model finally selected in R1 clearly demonstrates a good fit. Though this also applies to the Models 5 and 6, Model 7 was eventually selected because of the nature of its pathways. Particularly, the pathway from suspicion (SUS) to

Table 2
Model Building and Model Fit in the Samples R1, R2, and in the Total Sample

Sample	No.	Additional pathways	χ^2	df	RMSEA	SRMR	CFI
R1	1	(Figure 1 model)	288.00	53	0.11	0.083	0.91
	2	= 1 + EGC \rightarrow FTW	266.36	52	0.11	0.078	0.92
	3	= 2 + SAN \rightarrow CDR	223.35	51	0.096	0.069	0.94
	4	= 3 + FTW \rightarrow DET	175.18	50	0.082	0.061	0.95
	5	= 4 + SAN \rightarrow ALN	160.59	49	0.078	0.053	0.96
	6	= 5 + SAN \rightarrow APA	150.29	48	0.076	0.049	0.96
	7	= 6 + SUS \rightarrow EGC (Selected model)	141.63	47	0.074	0.048	0.96
R2	8	(Definitive model)	118.03	47	0.065	0.036	0.97
Total	9	(Definitive model)	183.82	47	0.063	0.037	0.97

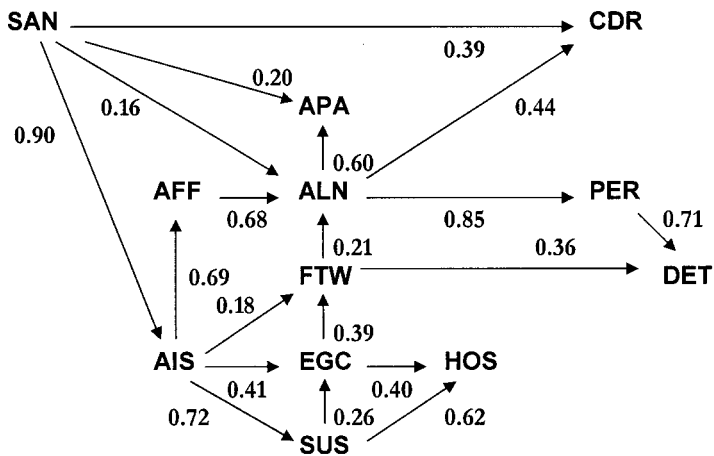
egocentricity (EGC) was added to causally connect a set of features (SUS, EGC, FTW, and DET) that already in nuce may represent paranoid schizophrenia with its emphasis on a delusional framework, reading personal significance into seemingly trivial activities of other people. With this addition, the model more properly incorporates the paranoid–nonparanoid distinction that has been found helpful in schizophrenia research (e.g., Goldstein, Held, & Cromwell, 1968). Of the remaining five newly discovered pathways in this model, the pathways from egocentrism (EGC) to living in a fantasy world (FTW), and from fantasy world (FTW) to delusional thinking (DET), form part of the causal chains just mentioned. Moreover, these pathways, as well as those that run from social anxiety (SAN) to, respectively, apathy (APA), cognitive derailment (CDR), and feelings of alienation (ALN), are almost self-explanatory, and hence, quite understandable from a causal point of view. In the top half of Figure 2, the finally selected model in R1 is portrayed, with the standardized coefficients associated with the causal pathways added to it. Except for the coefficient related to the pathway from AIS to FTW, that was significant at the 0.05 level, all coefficients proved to be at least significant at the 0.01 level.

Replication of the definite R1 model in sample R2, conducted on 362 subjects, resulted in a slightly better fit, as also shown in Table 2 (Model 8). Again, the CFI and SRMR values indicate a good fit, whereas the RMSEA value is just above Hu and Bentler's (1999) criterion value of 0.06. Applying the first of Hu and Bentler's joint criteria, the fit of the model appears adequate. Given these results, the same model as finally derived in R1 is depicted again in the bottom half of Figure 2, but now with the standardized structure coefficients as found in R2 included. All pathway coefficients proved to be at least significant at the 0.01 level, except for the coefficient related to the pathway from SAN to ALN, that was significant at the 0.05 level.

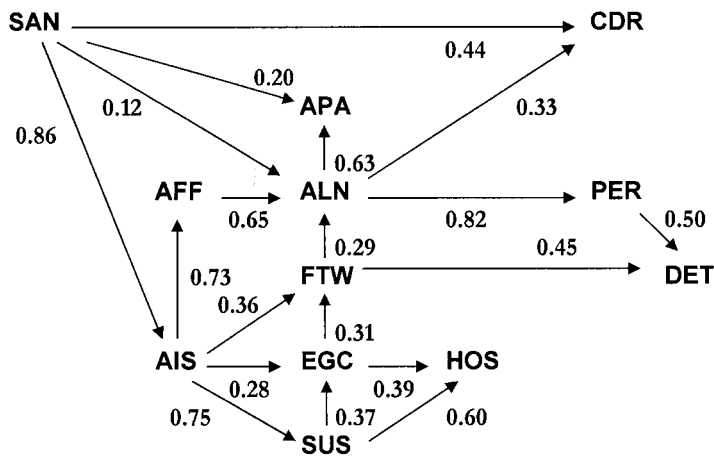
A two-sample analysis using R1 and R2 to test the invariance of the BETA matrix resulted in an overall χ^2 (with $df = 113$) of 276.70. As the sum of the χ^2 values separately obtained in the subsamples R1 and R2 amounts to 259.66 (sum $df = 94$), the null hypothesis of no invariance could not be rejected ($\chi^2_{\text{diff}} = 17.04$, $df = 19$, $p > 0.05$). Hence, no differences between the standardized pathway coefficients in the samples R1 and R2 could be observed.

For the sake of completeness, the LISREL testing of the finally selected model was also executed in the total sample, in this case comprising 733 (of the 771 HGA) subjects. As could be expected, given the above-mentioned findings, almost identical results

SAMPLE R1:



SAMPLE R2:



stages: 1 2 3 4

Figure 2. The selected SSQ model in samples R1 and R2. SAN = social anxiety; AIS = active isolation; AFF = affective flattening; APA = apathy; ALN = alienation; FTW = fantasy world; EGC = egocentrism; SUS = suspicion; HOS = hostility; CDR = cognitive derailment; PER = perceptual disturbances; DET = delusional thinking.

were obtained, with CFI = 0.97, RMSEA = 0.063, and SRMR = 0.037 (see Table 2).¹ Due to the large sample size, all standardized pathway coefficients appeared to be significantly different from zero, even at the 0.001 level. The model itself, inclusive of its pathway coefficients, is depicted in Figure 3.

¹Essentially, the same results as obtained in R1, R2, and the total HGA sample were also found in the sub-samples females ($n = 432$), males ($n = 269$), relatively young (age 16–35; $n = 371$), and relatively old subjects (age 36–59; $n = 330$). However, avoiding the risk of overtesting data obtained in dependent samples, these results will not be reported here.

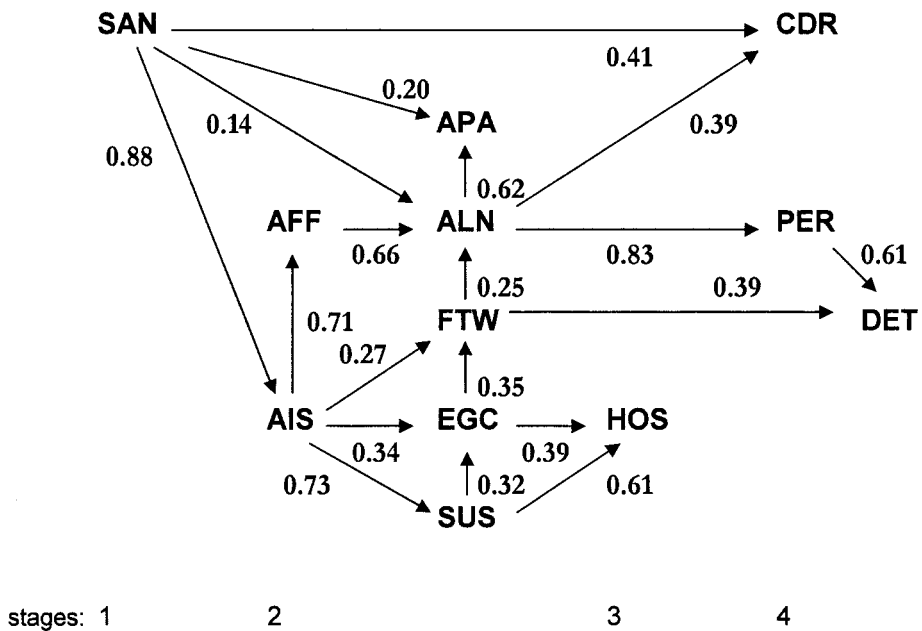


Figure 3. The selected SSQ model in the total sample. SAN = social anxiety; AIS = active isolation; AFF = affective flattening; APA = apathy; ALN = alienation; FTW = fantasy world; EGC = egocentrism; SUS = suspicion; HOS = hostility; CDR = cognitive derailment; PER = perceptual disturbances; DET = delusional thinking.

Given the relative complexity of the model (direct and indirect pathways), the standardized total effects of the latent variables on each other also are presented. These data are given in Table 3, as well as the variance percentages (squared multiple correlations) in the latent dependent variables (every variable but SAN) accounted for by the latent

Table 3

Squared Multiple Correlations for Structural Equations (R^2) and Standardized Total Effects of the Latent Variables in the SSQ Model

	R^2	SAN	AIS	CDR	AFF	PER	FTW	SUS	APA	ALN	DET	EGC	HOS
AIS	0.78	0.88											
CDR	0.53	0.67	0.23		0.26		0.10	0.01		0.39		0.03	
AFF	0.50	0.63	0.71										
PER	0.69	0.55	0.49		0.55		0.21	0.02		0.83		0.07	
FTW	0.30	0.42	0.47					0.11				0.35	
SUS	0.54	0.65	0.73										
APA	0.58	0.61	0.36		0.41		0.15	0.02		0.62		0.05	
ALN	0.78	0.66	0.59		0.66		0.25	0.03				0.09	
DET	0.74	0.50	0.49		0.34	0.61	0.52	0.06		0.51		0.18	
EGC	0.38	0.51	0.57					0.32					
HOS	0.80	0.60	0.67					0.74				0.39	

Note. SAN = social anxiety; AIS = active isolation; CDR = cognitive derailment; AFF = affective flattening; PER = perceptual disturbances; FTW = fantasy world; SUS = suspicion; APA = apathy; ALN = alienation; DET = delusional thinking; EGC = egocentrism; HOS = hostility.

independent variables. Of course, the standardized indirect effects may be obtained by subtracting the pathway coefficient values as displayed in Figure 3 from the total effects.

Discussion

The eventually selected and LISREL-tested model—the SSQ model—about the unfolding of the schizophrenic prodrome in a random sample (R1) of 375 subjects (drawn from a community-based sample [HGA] of 771 subjects), behaved very well after replication in a second random sample (R2) of 362 subjects that was also drawn from the total HGA sample. Not only did this model provide a satisfactory fit to the data in both samples, but it was also found that the structure coefficients associated with the pathways representing the model's postulated cause–effect relationships departed significantly from zero. After demonstrating the invariance of the structure coefficients in R1 and R2, the SSQ model was also tested in the total HGA sample. In this sample, as expected, the fit indices also indicated the model's ability to reproduce the observed variance–covariance matrix. Moreover, all pathway coefficients again differed significantly from zero. Given the observation (not reported in the present article) that the SSQ scales AFF, PER, APA, ALN, and DET turned out to be highly skewed (skew values ≥ 1), the model can be expected to fit the data even better than indicated by the fit values mentioned above (see, e.g., Satorra & Bentler, 1994). Of course, despite these positive results, other SSQ-based models describing the unfolding of the schizophrenic prodrome may outperform the model presented here, or at least fit the data equally well. However, approximately 50% of the pathways in the SSQ model were based on the existing clinical literature (e.g., Bleuler, 1911; Kretschmer, 1942; Livesley et al., 1998; Millon & Davis, 1996); thus, the model is plausible. Moreover, the model's plausibility is strengthened by the fact that the causal pathways in the SSQ model agree with the observation (e.g., Häfner et al., 1995; Yung & McGorry, 1996a) that negative symptoms precede the onset of positive or psychotic-like ones and that these pathways—at least in broad outline—span the various onset stages prior to schizophrenic psychosis as described by Docherty et al. (1978). Hence, the model as presently stated offers a dependable description of at least the major pathways in the temporal unfolding of the schizophrenic prodrome. However, admitting that an accepted model in covariance structure analysis is actually only “not-disconfirmed,” the final proof of the model's plausibility must be found in a retrospective investigation of a group of *DSM-IV* (APA, 1994) or *ICD-10* (WHO, 1992) diagnosed schizophrenic patients with a first psychotic episode. Particularly those patients that conform strongly to Kraepelin's (1913) definition of dementia praecox are likely to be characterized by the symptomatic pathways postulated in the SSQ model (see below).²

Defining the SSQ model as a model of prepsychotic disturbance (but see below), a transition to psychosis seems particularly dependent on highly elevated scores on PER (perceptual disturbances) and DET (delusional thinking), as the two SSQ scales probably most predictive with respect to the emergence of the principal psychotic symptoms, hallucinations and delusions. However, because nearly all pathways in the SSQ model ultimately lead to PER and DET, a general elevation on the scales of the SSQ also must enhance the probability of a transition to psychosis. It is from this perspective, that the process of prodromal unfolding as depicted by the SSQ model is a process of *positivation*

²Naturally, not all patients can be expected to have manifested all symptoms and/or their temporal arrangements as depicted in the SSQ model. Therefore, a measure must be applied that essentially refers to the number of SSQ pathways “walked on” prior to psychosis, corrected for the number of missing pathways due to the absence of SSQ symptoms.

(in Dutch: *positivering*). This process is assumed to rest on genetic influences that manifest themselves in negative symptoms (particularly social anxiety and active isolation) that, in turn, constitute a fertile soil for the development of antisocial, psychotic-like, and, finally, psychotic symptoms that appear to reflect the genetic background only obliquely or not at all. Therefore, admitting that negative and positive symptoms may statistically be interrelated (see above), Häfner and Maurer's (1991) notion of a common etiological factor lying behind both kinds of symptoms was rejected. However, as high SSQ scores may also be found in conditions (in particular, schizotypal and paranoid personality disorder) that usually do not progress to psychosis (see below), other factors, probably even more strongly influencing the likelihood of a psychotic transition, must also play an important or even decisive role. In this respect, the experience of an insoluble impasse (not unlikely associated with the occurrence of relational and occupational challenges in adolescence) noted by Docherty et al. (1978), cannabis use (e.g., Smit, Bolier, & Cuijpers, 2004), and the presence of childhood traumata may be mentioned, because these factors have been found not only in schizophrenia (Bleuler, 1972; Ross, Anderson, & Clark, 1994), but also in other diseases (see, e.g., Chu & Dill, 1990; Irwin, 2001; Putnam, Guroff, Silberman, Barban, & Post, 1986), to predict the development of psychotic symptoms.

Another issue relates to the meaning and concept of the schizophrenic prodrome itself. As already alluded to in the Introduction, the term prodrome is sometimes replaced by the term *at-risk mental state* (McGorry & Singh, 1995) to indicate that the symptoms included in that state do not necessarily progress to schizophrenic psychosis. Accordingly, the term *prodrome*, or the equivalent *precursor signs and symptoms* (Eaton, Badawi, & Melton, 1995), is actually a misnomer, and we have only stuck to this term because of its widespread use. Besides, any emphasis—as is the case, for instance, in the *DSM-IV* (APA, 1994) and *ICD-10* (WHO, 1992)—on psychotic phenomena as the principal symptoms of schizophrenia is usually misplaced (but see Kendler, 1986), because of their demonstrated nonpathognomonic status (e.g., Peralta & Cuesta, 1999), their relatively weak or even absent relationship with schizophrenia's pathophysiology and genetic background (Tsuang et al., 2000), and especially because many precursor symptoms actually coincide with what Kraepelin (1913) and Bleuler (1911) indicated as the most defining characteristics or Grundsymptome of schizophrenia, which may or may not precede the later development of psychotic and other accessory symptoms. Seen from this point of view, the schizophrenic prodrome seems better regarded as a nonpsychotic manifestation of the same condition that, if also characterized by psychotic features, is typically diagnosed—particularly in more recent times—as schizophrenia. Hence, the set of SSQ symptoms actually seems to indicate a *minor* form of schizophrenia.

With the SSQ model now established a more valid definition of schizophrenia as a psychotic disease can be developed. Indeed, if the prodromal pathways may find their ultimate endpoint in Stage 4 (psychotic disorganization) and Stage 5 (psychotic resolution) in Docherty et al.'s (1978) scheme, the psychotic form of schizophrenia seems nothing but the equivalent of the schizophrenic prodrome with psychosis. To present a SSQ related description of this *major* or full-blown psychotic form of schizophrenia, several pathways to the SSQ model need to be added, not only (see above) a path from PER (perceptual disturbances) to hallucinations and a path from DET (delusional thinking) to delusions, but also a pathway from CDR (cognitive derailment) to formal thought disorder (e.g., incoherence and the use of neologisms). The last-mentioned pathway is based on our already stated assumption that the causal origins of incoherence and other formal thought disturbances lie in vague and impoverished thinking. However, it is important to realize that the resultant definition of the full-blown SSQ form of schizophrenia may only indicate a subtype of what is usually rubricated under the term

schizophrenia. Indeed, what is presently or formerly called schizophrenia refers to such widely different manifestations as Kasanin's (1933) schizoaffective disorder, Vaillant's (1964) good prognosis schizophrenia, and Hoch and Polatin's (1949) pseudo-neurotic schizophrenia, as well as to the simple and latent schizophrenias described by Bleuler (1911), whereas in more present-day definitions—following, for instance, the decisions in *DSM-III* (APA, 1980)—much narrower boundaries are emphasized, in combination with a greater accent on Schneiderian first-rank symptoms (Andreasen, 1987; Andreasen & Flaum, 1991). Even with the advent of the classificatory schemes *DSM-IV* (APA, 1994) and *ICD-10* (WHO, 1992), the controversies swirling about the boundaries of the concept have not ended, because *ICD-10* handles a somewhat broader definition of schizophrenia, usually associated with better prognosis. Hence, the group of patients formerly or presently diagnosed with schizophrenia denotes a relatively heterogeneous group (Vlaminck, 2002); it is why several investigators have tried to delineate more homogeneous subgroups, for instance, on the basis of severity or outcome (see Roy, Merette, & Maziade, 2001). In defining the core of schizophrenia according to the SSQ model, our own delineation of schizophrenia, whether minor or major, has one great advantage: it is a subtype of schizophrenia that evidently goes back to the original characterization of dementia praecox or schizophrenia by Kraepelin and Bleuler. As such, there is every reason to stress our definition of schizophrenia in favor of other former or present-day definitions that more or less grossly deviate from these original characterizations.

A partly similar redefinition of the concept of schizophrenia has been proposed by Tsuang and collaborators (e.g., Tsuang et al., 2000; Faraone et al., 2001). After defining the central features of schizophrenia as a constellation of negative symptoms, psychosocial dysfunction, brain abnormalities, and neuropsychological deficits, which are together termed *schizotaxia*, the psychotic form of schizophrenia is described as *schizotaxia with psychosis* (Tsuang et al., 2000, p. 1048). However, a major problem with this redefinition lies in the fact that the schizotaxia concept merely embraces negative symptoms, which was done because these features, as well as the cited biological factors, have been found to particularly reflect the genetic influences that contribute to schizophrenia. Whatever the merits of this reformulation from a genetic point of view, the SSQ model makes clear that there are developmental pathways according to which negative symptoms, partly via the emergence of asocial features, are linked with psychotic-like features, which seem likely to precede the eventual development of full-blown psychotic phenomena. Furthermore, the set of symptoms in the SSQ model—although relatively small—represents the total domain of prodromal symptoms as specified by Yung and McGorry (1996a) almost exhaustively (see above), and, hence, by equating the SSQ representation with the minor form of schizophrenia, the redefinition of the psychotic form of schizophrenia as *the SSQ model with psychosis* appears to describe this condition in a satisfactory manner.

The redefinition of schizophrenia according to the SSQ model also is important given the present-day discussion concerning the tenability of the schizophrenia concept. If faith were placed in Boyle's (1990) arguments, Kraepelin would never had observed any set of regularities for inferring dementia praecox or schizophrenia. From a similar stance, Bentall, Jackson, and Pilgrim (1988) urged the abandonment of the concept of schizophrenia altogether, pursuing instead the study of various individual symptoms separately. However, based on the SSQ evidence mentioned above, there is little reason to discard the notion of a symptom complex. Rather, a proper definition of schizophrenia, as grounded in the SSQ model, can be said to refer to a distinct cluster of mutually associated and causally dependent symptoms. Because these features are actually continua, the SSQ model, with or without psychotic extensions, denotes at the same time a dimensional representation of schizophrenia.

Finally, in addition to an extension to psychosis, the SSQ model might also be widened in the direction of childhood behavioral precursors and premorbid personality factors. As it is clear that the initial symptoms in the SSQ model are negative features, this widening particularly relates to those premorbid features and influences that have been found associated with the occurrence of these symptoms. As such, prominence must be given to poor premorbid social and occupational adjustment (Andreasen & Olsen, 1982; Walker & Lewine, 1988), premorbid schizoid personality (Cuesta et al., 2002), and negative-type childhood problems, like passivity, shyness, and social withdrawal (Baum & Walker, 1995; Cannon, Mednick & Parnas, 1990). Normal personality traits like (high) neuroticism and (low) extraversion (Ross, Lutz, & Bailey, 2002; Van Kampen, 1999) must also be considered. This further extension seems fully reminiscent of Kraepelin's (1913) description of dementia praecox (see, e.g., for the childhood precursors: Kraepelin, 1913, p. 922), and, hence, corroborates the view that the SSQ model, with or without psychotic extensions, agrees well with Kraepelin's original characterization of that disorder. This view is strengthened by the fact that the negative symptoms present in the SSQ model have also been found to be rather strongly associated with such Kraepelinian features more or less typical for dementia praecox as an early and insidious onset (Fenton & McGlashan, 1991; Gupta, Rajaprabhakaran, Arndt, Flaum, & Andreasen, 1995), a more enduring course (Gupta et al., 1997), and poor outcome (Addington, Van Mastrigt, & Addington, 2003). A somewhat weaker agreement is apparent with regard to Bleuler's (1911) conceptualization of schizophrenia, though Bleuler too noted several of the above-mentioned characteristics, such as a lack of interest in the patient's social environment in childhood, a gradual onset, and a long-term course.

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